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L10 and diagnosis	27

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 Derwent World Patents Index
 IBM Technical Disclosure Bulletins

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L10 and diagnosis

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USPT,PGPB,JPAB,EPAB,DWPI,TDBD	L10 and diagnosis	27	<u>L11</u>
USPT,PGPB,JPAB,EPAB,DWPI,TDBD	L8 and diabetes	61	<u>L10</u>
USPT,PGPB,JPAB,EPAB,DWPI,TDBD	L8 and diabet?	0	<u>L9</u>
USPT,PGPB,JPAB,EPAB,DWPI,TDBD	L3 and insulin	133	<u>L8</u>
USPT,PGPB,JPAB,EPAB,DWPI,TDBD	L3 and insulin?	0	<u>L7</u>
USPT,PGPB,JPAB,EPAB,DWPI,TDBD	L3 and (insulin? or diabet?)	0	<u>L6</u>
USPT,PGPB,JPAB,EPAB,DWPI,TDBD	keratinocyte? and (FABP?)	9	<u>L5</u>
USPT,PGPB,JPAB,EPAB,DWPI,TDBD	keratinocyte? and (FABP)	11	<u>L4</u>
USPT,PGPB,JPAB,EPAB,DWPI,TDBD	keratinocyte? and (fatty adj acid?)	491	<u>L3</u>
USPT,PGPB,JPAB,EPAB,DWPI,TDBD	Mal1?	3	<u>L2</u>
USPT,PGPB,JPAB,EPAB,DWPI,TDBD	Mal1	12	<u>L1</u>



Day : Monday
Date: 10/15/2001
Time: 14:39:37

Inventor Name Search

Enter the **first few letters** of the Inventor's Last Name.
Additionally, enter the **first few letters** of the Inventor's First name.

Last Name

First Name

hotamisligil	gokhan	Search
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AND CURRENT DISCOVER FILE IS DATED 07 AUGUST 2001

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=> s Mall
L1 259 MAL1

=> s (keratinocyte adj fatty adj acid)
L2 0 (KERATINOCYTE ADJ FATTY ADJ ACID)

=> s keratinocyte
L3 76939 KERATINOCYTE

=> s l3 and (fatty adj acid?)
L4 0 L3 AND (FATTY ADJ ACID?)

=> s l3 and FABP?
L5 129 L3 AND FABP?

=> s L3 and (binding adj protein?)
5 FILES SEARCHED...
L6 0 L3 AND (BINDING ADJ PROTEIN?)

=> s l1 and (insulin? or diabet?)
L7 2 L1 AND (INSULIN? OR DIABET?)

=> s L5 and (insulin? or diabet?)
L8 9 L5 AND (INSULIN? OR DIABET?)

=> s l7 or l8
L9 10 L7 OR L8

=> dup rem l9
DUPLICATE IS NOT AVAILABLE IN 'REGISTRY'.
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L10 6 DUP REM L9 (4 DUPLICATES REMOVED)

=> d l10 ibib abs 1-
YOU HAVE REQUESTED DATA FROM 6 ANSWERS - CONTINUE? Y/(N):y

L10 ANSWER 1 OF 6 CAPLUS COPYRIGHT 2001 ACS

ACCESSION NUMBER: 2001:617839 CAPLUS
 DOCUMENT NUMBER: 135:200396
 TITLE: Treatment lipid metabolic disorders by inhibiting **Mall**, the keratinocyte lipid binding protein activity
 INVENTOR(S): Hotamisligil, Gokhan S.
 PATENT ASSIGNEE(S): President and Fellows of Harvard College, USA
 SOURCE: PCT Int. Appl., 27 pp.
 CODEN: PIXXD2
 DOCUMENT TYPE: Patent
 LANGUAGE: English
 FAMILY ACC. NUM. COUNT: 1
 PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
-----	----	-----	-----	-----
WO 2001060384	A1	20010823	WO 2001-US5019	20010216
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
PRIORITY APPLN. INFO.:			US 2000-183106	P 20000217
AB The invention is based on the discovery that decreasing Mall (also called keratinocyte fatty acid binding protein) expression prevents or inhibits the development of obesity, insulin resistance, diabetes , dyslipidemia, and atherosclerosis. Accordingly, the invention features a method of preventing or inhibiting such conditions by administering to a mammal, who has been identified as suffering from or at risk of developing one or more of the above-listed pathologies, a compd. that reduces expression or activity of Mall . Preferably, the compd. inhibits transcription of endogenous Mall by binding to a cis-acting regulatory sequence of the Mall gene and decreases Mall transcription. Alternatively, the compd. inhibits translation of Mall mRNA into a Mall gene product by antisense therapy carried out by administering a single stranded nucleic acid complementary at least a portion of Mall mRNA. The invention also includes a method of preventing or inhibiting the development of obesity, insulin resistance, diabetes , dyslipidemia, and atherosclerosis by administering to a mammal a compd. that reduces activity of Mall . The invention also includes a method of diagnosing individuals who are at risk of developing lipid metabolic disorders.				
REFERENCE COUNT:		6		
REFERENCE(S):		(1) Hertzfel, A; GENE 1998, V221(2), P235 CAPLUS (2) Hotamisligil, G; WO 0047734 A 2000 CAPLUS (3) Hotamisligil, G; SCIENCE 1996, V274(5291), P1377 CAPLUS (4) Kane, C; BIOCHEMISTRY 1996, V35(9), P2894 CAPLUS (6) Shaughnessy, S; DIABETES 2000, V49(6), P904		
CAPLUS				

ALL CITATIONS AVAILABLE IN THE RE FORMAT

ACCESSION NUMBER: 2001103644 MEDLINE
DOCUMENT NUMBER: 20515662 PubMed ID: 11060343
TITLE: Lipid-binding proteins modulate ligand-dependent trans-activation by peroxisome proliferator-activated receptors and localize to the nucleus as well as the cytoplasm.
AUTHOR: Helledie T; Antonius M; Sorensen R V; Hertzelt A V; Bernlohr
CORPORATE SOURCE: D A; Kolvraa S; Kristiansen K; Mandrup S
Department of Molecular Biology, University of Southern Denmark, Odense, DK-5230 Odense M, Denmark.
SOURCE: JOURNAL OF LIPID RESEARCH, (2000 Nov) 41 (11) 1740-51.
Journal code: IX3. ISSN: 0022-2275.
PUB. COUNTRY: United States
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200102
ENTRY DATE: Entered STN: 20010322
Last Updated on STN: 20010322
Entered Medline: 20010208

AB Peroxisome proliferator-activated receptors (PPARs) are activated by a variety of fatty acids, eicosanoids, and hypolipidemic and **insulin**-sensitizing drugs. Many of these compounds bind avidly to members of a family of small lipid-binding proteins, the fatty acid-binding proteins (**FABPs**). Fatty acids are activated to CoA esters, which bind with high affinity to the acyl-CoA-binding protein (ACBP). Thus, the availability of known and potential PPAR ligands may be regulated by lipid-binding proteins. In this report we show by transient transfection of CV-1 cells that coexpression of ACBP and adipocyte lipid-binding protein (ALBP) exerts a ligand- and PPAR subtype-specific attenuation of PPAR-mediated trans-activation, suggesting that lipid-binding proteins, when expressed at high levels, may function as negative regulators of

PPAR activation by certain ligands. Expression of ACBP, ALBP, and **keratinocyte** lipid-binding protein (KLBP) is induced during adipocyte differentiation, a process during which PPARgamma plays a prominent role. We present evidence that endogenous ACBP, ALBP, and KLBP not only localize to the cytoplasm but also exhibit a prominent nuclear localization in 3T3-L1 adipocytes. In addition, forced expression of

ACBP, ALBP, and KLBP in CV-1 cells resulted in a substantial accumulation of all

three proteins in the nucleus. These results suggest that lipid-binding proteins, contrary to the general assumption, may exert their action in the nucleus as well as in the cytoplasm.

L10 ANSWER 3 OF 6 CAPLUS COPYRIGHT 2001 ACS

ACCESSION NUMBER: 2000:647848 CAPLUS
DOCUMENT NUMBER: 134:351081
TITLE: Adipocyte metabolism in adipocyte fatty acid binding protein knockout (aP2-/-) mice after short-term high-fat feeding: functional compensation by the **keratinocyte** [sic] fatty acid binding protein. [Erratum to document cited in CA133:132923]
AUTHOR(S): Shaughnessy, Sara; Smith, Elizabeth R.; Kodukula, Sarala; Storch, Judith; Fried, Susan K.
CORPORATE SOURCE: Department of Nutritional Sciences, Rutgers University, New Brunswick, NJ, 08901-8525, USA
SOURCE: Diabetes (2000), 49(9), 1617

CODEN: DIAEAZ; ISSN: 0012-1797
PUBLISHER: American Diabetes Association
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The word "**keratinocyte**" was incorrectly spelled as
"keritinocyte".
REFERENCE COUNT: 3
REFERENCE(S): (1) Mammes, O; Diabetes 1998, V47, P487 CAPLUS
(2) Shaughnessy, S; Diabetes 2000, V49, P904 CAPLUS
(3) Virtanen, S; Diabetes 2000, V49, P912 CAPLUS

L10 ANSWER 4 OF 6 SCISEARCH COPYRIGHT 2001 ISI (R)

ACCESSION NUMBER: 2000:564381 SCISEARCH

THE GENUINE ARTICLE: 335GZ

TITLE: Adenovirus-mediated gene transfer in primary murine
adipocytes

AUTHOR: Hertz A V; Sanders M A; Bernlohr D A (Reprint)

CORPORATE SOURCE: UNIV MINNESOTA, DEPT BIOCHEM MOL BIOL & BIOPHYS, ST PAUL,
MN 55108 (Reprint); UNIV MINNESOTA, DEPT BIOCHEM MOL BIOL
& BIOPHYS, ST PAUL, MN 55108; UNIV MINNESOTA, IMAGING

CTR,
ST PAUL, MN 55108

COUNTRY OF AUTHOR: USA

SOURCE: JOURNAL OF LIPID RESEARCH, (JUL 2000) Vol. 41, No. 7, pp.
1082-1086.

Publisher: LIPID RESEARCH INC, 9650 ROCKVILLE PIKE,
BETHESDA, MD 20814-3998.

ISSN: 0022-2275.

DOCUMENT TYPE: Article; Journal

FILE SEGMENT: LIFE

LANGUAGE: English

REFERENCE COUNT: 11

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB The transfer of genes into primary murine adipocytes using an
adenovirus system has been developed. A recombinant adenovirus was
constructed (expressing green fluorescent protein [GFP] under the control
of the strong cytomegalovirus [CMV] promoter and a luciferase reporter
gene under the control of the weak adipocyte promoter **keratinocyte**
lipid-binding protein [KLBP/**FABP5**]) and incubated with primary
adipocytes from C57BL/6J mice. Analysis of infected cells by confocal
microscopy detected GFP expression in both the cytoplasm and nucleus of
adipocytes with a 64% efficiency of infection. To demonstrate the
applicability of this method in the study of gene regulation,
adenovirus-infected adipocytes exhibited significant levels of luciferase
activity even from a weak promoter. TPA treatment of infected adipocytes
increased luciferase activity, consistent with previous studies
indicating
that the KLBP/ **FABP5** gene is up-regulated by phorbol esters.
These results provide an efficient, convenient, and sensitive method to
transiently infect primary murine adipocytes, facilitating protein
expression or permitting analysis of reporter gene activity from both
viral and endogenous promoters.

L10 ANSWER 5 OF 6 CAPLUS COPYRIGHT 2001 ACS

ACCESSION NUMBER: 2000:398680 CAPLUS

DOCUMENT NUMBER: 133:132923

TITLE: Adipocyte metabolism in adipocyte fatty acid binding
protein knockout (aP2-/-) mice after short-term
high-fat feeding: functional compensation by the
keratinocyte fatty acid binding protein

AUTHOR(S): Shaughnessy, Sara; Smith, Elizabeth R.; Kodukula, Sarala; Storch, Judith; Fried, Susan K.
CORPORATE SOURCE: Department of Nutritional Sciences, Rutgers University, New Brunswick, NJ, 08901-8525, USA
SOURCE: Diabetes (2000), 49(6), 904-911
CODEN: DIAEAZ; ISSN: 0012-1797
PUBLISHER: American Diabetes Association
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Mice null for adipocyte fatty acid binding protein (AFABP) compensate by increasing expression of **keratinocyte** fatty acid binding protein (KFABP). In the present study, AFABP knockout (KO) and wild-type (WT) mice became equally obese on a high-fat diet, as judged by fat pad wts., adipocyte size, and body compn. anal. High-fat feeding led to moderate **insulin** resistance in both WT and AFABP knockout mice, as indicated by an .apprx.2-fold increase in plasma **insulin**. However, in the high fat-fed mice, plasma glucose levels were .apprx.15% lower in the AFABP-KO mice. Adipocytes isolated from AFABP-KO and WT

mice

fed high- or low-fat diets exhibited similar rates of basal and norepinephrine-stimulated lipolysis and **insulin**-stimulated rates of glucose conversion to fatty acids and glyceride-glycerol. However, basal glucose conversion to fatty acids was higher in adipocytes of AFABP-KO mice. Adipocyte tumor necrosis factor-.alpha. release was similarly increased by high-fat diet-induced obesity in both WT and AFABP-KO mice. As assessed by Western blot anal., the level of KFABP protein in AFABP-KOs was .apprx.40% of the level of AFABP in WT controls. The binding affinities of KFABP for long-chain fatty acids were 2- to 4-fold higher than those of AFABP, but the relative affinities for different fatty acids were similar. As for AFABP, the rate of fatty acid transfer from KFABP to model phospholipid vesicles was increased with acceptor membrane concn. and by inclusion of acidic phospholipids, indicating a similar mechanism of transfer. We conclude KFABP can functionally compensate for the absence of AFABP, resulting in no major alterations in adipocyte metab. or fat accumulation in response to short-term feeding of high-fat diets that result in moderate hyperinsulinemia.

REFERENCE COUNT: 24

REFERENCE(S): (1) Binas, B; FASEB J 1999, V13, P805 CAPLUS
(2) Coe, N; J Lipid Res 1999, V40, P967 CAPLUS
(3) DiGirolamo, M; J Lipid Res 1974, V15, P332 CAPLUS
(4) Dole, V; J Biol Chem 1960, V235, P2595 CAPLUS
(5) Edens, N; Am J Physiol 1993, V265, PE374 CAPLUS
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L10 ANSWER 6 OF 6 EMBASE COPYRIGHT 2001 ELSEVIER SCI. B.V.

ACCESSION NUMBER: 94371631 EMBASE

DOCUMENT NUMBER: 1994371631

TITLE: Retinoic acid induces expression of PA-**FABP**
(psoriasis-associated fatty acid-binding protein) gene in human skin in vivo but not in cultured skin cells.

AUTHOR: Larsen F.G.; Voorhees J.J.; Astrom A.

CORPORATE SOURCE: Department of Dermatology, Bispebjerg Hospital, 2400 Copenhagen NV, Denmark

SOURCE: Experimental Dermatology, (1994) 3/5 (212-218).

ISSN: 0906-6705 CODEN: EXDEEY

COUNTRY: Denmark

DOCUMENT TYPE: Journal; Article

FILE SEGMENT: 013 Dermatology and Venereology
037 Drug Literature Index

LANGUAGE: English

SUMMARY LANGUAGE: English

AB PA-**FABP** (psoriasis-associated fatty acid-binding protein) is a new member of a group of low-molecular-weight proteins that are highly up-regulated in psoriatic skin and that share similarity to fatty acid-binding proteins. In this study we demonstrate that PA-**FABP** transcripts are expressed in human skin in vivo and that topical application of 0.05% retinoic acid (RA) cream results in a rapid induction

of PA-**FABP** transcripts following treatment for 16 hours and persists at increasing levels after 48 and 96 h of RA treatment. The PA-**FABP** mRNA response to RA was reduced by approximately 50% when patients concurrently were treated with RA and 0.025% clobetasol propionate (CLO) for 48 and 96 h, whereas treatment with CLO alone resulted in PA-**FABP** transcript levels not significantly different from vehicle-treated skin. When comparing the effects of a well-known irritant, sodium lauryl sulfate (SLS), to those of RA and its vehicle, 0.05% RA cream but not 2% SLS in RA vehicle caused PA-**FABP** mRNA induction after 16 h. SLS treatment of human skin for 96 h caused a slight increase in PA-**FABP** transcripts, but markedly less than that observed in response to RA treatment. Incubation of cultured human **keratinocytes** or skin fibroblasts with RA for up to 48 h did not significantly induce PA-**FABP** transcripts. Expression of PA-**FABP** message in **keratinocytes** was observed to be induced by calcium and fetal calf serum (FCS), while tetra-decanoyl phorbol acetate (TPA) caused little or no induction. Taken together, the marked inducibility of the PA-**FABP** gene is compatible with the possibility that this gene might be important in RA-mediated regulation of human skin growth and differentiation.

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NEWS 5 Apr 23 Search Derwent WPINDEX by chemical structure
NEWS 6 Apr 23 PRE-1967 REFERENCES NOW SEARCHABLE IN CAPLUS AND CA
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to PHARMASEARCH
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NEWS 15 Oct 09 Number of Derwent World Patents Index updates increased
NEWS 16 Oct 15 Calculated properties now in the REGISTRY/ZREGISTRY File

NEWS EXPRESS August 15 CURRENT WINDOWS VERSION IS V6.0c,
CURRENT MACINTOSH VERSION IS V6.0 (ENG) AND V6.0J (JP),
AND CURRENT DISCOVER FILE IS DATED 07 AUGUST 2001

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=> s diabetes or (insulin adj resistance)
L1 592865 DIABETES OR (INSULIN ADJ RESISTANCE)

=> s l1 and diagnosis
L2 71359 L1 AND DIAGNOSIS

=> s l2 and transcript?
L3 336 L2 AND TRANSCRIPT?

=> s l2 and Mall
L4 1 L2 AND MAL1

=> s l2 and (keratinocyte adj fatty adj acid?)
5 FILES SEARCHED...
L5 0 L2 AND (KERATINOCYTE ADJ FATTY ADJ ACID?)

=> d l4 ibib abs 1-
YOU HAVE REQUESTED DATA FROM 1 ANSWERS - CONTINUE? Y/(N):y

L4 ANSWER 1 OF 1 CAPLUS COPYRIGHT 2001 ACS
ACCESSION NUMBER: 2001:617839 CAPLUS
DOCUMENT NUMBER: 135:200396
TITLE: Treatment lipid metabolic disorders by inhibiting
Mall, the keratinocyte lipid binding protein
activity
INVENTOR(S): Hotamisligil, Gokhan S.
PATENT ASSIGNEE(S): President and Fellows of Harvard College, USA
SOURCE: PCT Int. Appl., 27 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001060384	A1	20010823	WO 2001-US5019	20010216
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN,				

CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR,
 HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT,
 LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU,
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 BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

PRIORITY APPLN. INFO.: US 2000-183106 P 20000217

AB The invention is based on the discovery that decreasing **Mall**
 (also called keratinocyte fatty acid binding protein) expression prevents
 or inhibits the development of obesity, insulin resistance,
diabetes, dyslipidemia, and atherosclerosis. Accordingly, the
 invention features a method of preventing or inhibiting such conditions
 by administering to a mammal, who has been identified as suffering from or
 at risk of developing one or more of the above-listed pathologies, a compd.
 that reduces expression or activity of **Mall**. Preferably, the
 compd. inhibits transcription of endogenous **Mall** by binding to a
 cis-acting regulatory sequence of the **Mall** gene and decreases
Mall transcription. Alternatively, the compd. inhibits
 translation of **Mall** mRNA into a **Mall** gene product by
 antisense therapy carried out by administering a single stranded nucleic
 acid complementary at least a portion of **Mall** mRNA. The
 invention also includes a method of preventing or inhibiting the
 development of obesity, insulin resistance, **diabetes**,
 dyslipidemia, and atherosclerosis by administering to a mammal a compd.
 that reduces activity of **Mall**. The invention also includes a
 method of diagnosing individuals who are at risk of developing lipid
 metabolic disorders.

REFERENCE COUNT: 6

REFERENCE(S): (1) Hertz, A; GENE 1998, V221(2), P235 CAPLUS
 (2) Hotamisligil, G; WO 0047734 A 2000 CAPLUS
 (3) Hotamisligil, G; SCIENCE 1996, V274(5291), P1377
 CAPLUS
 (4) Kane, C; BIOCHEMISTRY 1996, V35(9), P2894 CAPLUS
 (6) Shaughnessy, S; DIABETES 2000, V49(6), P904

CAPLUS

ALL CITATIONS AVAILABLE IN THE RE FORMAT

=>

---Logging off of STN---

=>

Executing the logoff script...

=> LOG Y

COST IN U.S. DOLLARS

SINCE FILE	TOTAL
ENTRY	SESSION
70.18	70.33

FULL ESTIMATED COST

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)

SINCE FILE	TOTAL
ENTRY	SESSION

CA SUBSCRIBER PRICE

-0.59

-0.59

* * * * *

Dear valued customer,

Your feedback is important to us. Would you kindly take a moment to complete our survey? This survey will only take about 5-10 minutes to complete. Your responses will be kept confidential and will help us improve STN Express with Discover! for your future use. Please click on the following link to access the survey.

<http://www.cas.org/ONLINE/STN/ExpressSurveyForm.html?LOGINID=SSSPTA1635LAN>

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STN INTERNATIONAL LOGOFF AT 15:13:21 ON 15 OCT 2001